Subject Search


```
=> FILE CANCERLIT
FILE 'CANCERLIT' ENTERED AT 17:11:48 ON 21 JUN 2002

 FILE COVERS 1963 TO 14 Jun 2001 (20010614/ED)

 CANCERLIT thesauri in the /CN, /CT, and /MN fields incorporate the
 MeSH 2000 vocabulary.  Enter HELP THESAURUS for details.

 This file contains CAS Registry Numbers for easy and accurate substance
 identification.
```

Cancer lit Tree # for antibodies (pre-explosion)

Point of Contact:
Thomas G. Larson, Ph.D.
703-308-7309
CM1, Rm. 6 B 01

```
=> D QUE L6
L1   (      2483) SEA FILE=CANCERLIT ABB=ON  PLU=ON  LAMININ/CT
L2   (    193246) SEA FILE=CANCERLIT ABB=ON  PLU=ON  CARCINOMA+NT/CT
L3   (     94758) SEA FILE=CANCERLIT ABB=ON  PLU=ON  D24.611.125./CT
L4   (     20853) SEA FILE=CANCERLIT ABB=ON  PLU=ON  L3 (L) (TU OR PD OR PK OR
                  AD)/CT
L5   (      1014) SEA FILE=CANCERLIT ABB=ON  PLU=ON  L1/MAJ
L6             6 SEA FILE=CANCERLIT ABB=ON  PLU=ON  L2 AND L4 AND L5

=> D QUE L11
L7   (    193246) SEA FILE=CANCERLIT ABB=ON  PLU=ON  CARCINOMA+NT/CT
L8   (     78567) SEA FILE=CANCERLIT ABB=ON  PLU=ON  L7 (L) TH/CT
L9   (      4588) SEA FILE=CANCERLIT ABB=ON  PLU=ON  KALININ OR MEROSIN OR
                  LAMININ
L10  (       313) SEA FILE=CANCERLIT ABB=ON  PLU=ON  L9 (3A) (ANTI OR ANTIBOD?
                  OR MAB OR IMMUNOGLOBULIN)
L11            2 SEA FILE=CANCERLIT ABB=ON  PLU=ON  L10 AND L8
```

Tu = Therapeutic Use
PD = Pharmacology
PK = Pharmaco-Kinetics
AD = Administration & dosage
Maj = Major topic of document
Th. = Therapy

```
=> S L6 OR L11
L165           8 L6 OR L11

=> FILE BIOSIS
FILE 'BIOSIS' ENTERED AT 17:12:18 ON 21 JUN 2002
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FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 19 June 2002 (20020619/ED)

=> D QUE L22
L12  (     14286) SEA FILE=BIOSIS ABB=ON  PLU=ON  KALININ# OR LAMININ# OR
                  MEROSIN# OR NICEIN#
L13  (      3272) SEA FILE=BIOSIS ABB=ON  PLU=ON  GAMMA2 OR GAMMA 2
L14  (    644158) SEA FILE=BIOSIS ABB=ON  PLU=ON  ANTIBOD? OR IMMUNOGLOB? OR IG#
                  OR MAB
L15  (    210831) SEA FILE=BIOSIS ABB=ON  PLU=ON  METASTA? OR INVASI? OR INVAD?
L16  (     17404) SEA FILE=BIOSIS ABB=ON  PLU=ON  L12 OR L13
L17  (      1388) SEA FILE=BIOSIS ABB=ON  PLU=ON  L16 (5A) L14
L18  (   2872541) SEA FILE=BIOSIS ABB=ON  PLU=ON  TREAT? OR INHIBIT? OR THERAP?
L19  (     26275) SEA FILE=BIOSIS ABB=ON  PLU=ON  L15 (5A) L18
L20  (        14) SEA FILE=BIOSIS ABB=ON  PLU=ON  L17 AND L19
L21  (         4) SEA FILE=BIOSIS ABB=ON  PLU=ON  L20 AND (ANTILAMININ OR
                  LAMININ-MONOCLONAL OR ANTI-LAMININ) (2W) ANTIBOD?
```

```
L22           3 SEA FILE=BIOSIS ABB=ON  PLU=ON  L21 NOT TRYPANOSOMA/TI


=> D QUE L32
L23 (     14286)SEA FILE=BIOSIS ABB=ON  PLU=ON  KALININ# OR LAMININ# OR
                MEROSIN# OR NICEIN#
L24 (      3272)SEA FILE=BIOSIS ABB=ON  PLU=ON  GAMMA2 OR GAMMA 2
L25 (    644158)SEA FILE=BIOSIS ABB=ON  PLU=ON  ANTIBOD? OR IMMUNOGLOB? OR IG#
                OR MAB
L26 (    210831)SEA FILE=BIOSIS ABB=ON  PLU=ON  METASTA? OR INVASI? OR INVAD?
L27 (     17404)SEA FILE=BIOSIS ABB=ON  PLU=ON  L23 OR L24
L28 (      1388)SEA FILE=BIOSIS ABB=ON  PLU=ON  L27 (5A) L25
L29 (   2872541)SEA FILE=BIOSIS ABB=ON  PLU=ON  TREAT? OR INHIBIT? OR THERAP?
L30 (     26275)SEA FILE=BIOSIS ABB=ON  PLU=ON  L26 (5A) L29
L31 (        14)SEA FILE=BIOSIS ABB=ON  PLU=ON  L28 AND L30
L32           1 SEA FILE=BIOSIS ABB=ON  PLU=ON  L31 AND ANTIBODY PERTURBATION


=> D QUE L40
L33 (     14286)SEA FILE=BIOSIS ABB=ON  PLU=ON  KALININ# OR LAMININ# OR
                MEROSIN# OR NICEIN#
L34 (      3272)SEA FILE=BIOSIS ABB=ON  PLU=ON  GAMMA2 OR GAMMA 2
L35 (        22)SEA FILE=BIOSIS ABB=ON  PLU=ON  ANTI (W) (GAMMA 2 OR GAMMA2)
L36 (    210831)SEA FILE=BIOSIS ABB=ON  PLU=ON  METASTA? OR INVASI? OR INVAD?
L37 (     17404)SEA FILE=BIOSIS ABB=ON  PLU=ON  L33 OR L34
L38 (   2872541)SEA FILE=BIOSIS ABB=ON  PLU=ON  TREAT? OR INHIBIT? OR THERAP?
L39 (     26275)SEA FILE=BIOSIS ABB=ON  PLU=ON  L36 (5A) L38
L40           0 SEA FILE=BIOSIS ABB=ON  PLU=ON  L35 AND L37 AND L39


=> D QUE L54
L41 (    293485)SEA FILE=BIOSIS ABB=ON  PLU=ON  ?CARCINOMA?
L42 (      5578)SEA FILE=BIOSIS ABB=ON  PLU=ON  (INTRAEPITHELIAL OR INTRA
                EPITHELIAL) (W) NEOPLAS? OR BOWEN? DISEASE OR BASAL (2A) NEVUS
                OR LINITIS PLASTICA
L43 (      3773)SEA FILE=BIOSIS ABB=ON  PLU=ON  (CARCINOID OR KLATSKIN? OR
                KRUKENBURG?) (1W) (TUMOR OR TUMOUR OR SYNDROME)
L44 (    299637)SEA FILE=BIOSIS ABB=ON  PLU=ON  (L41 OR L42 OR L43)
L45 (     14286)SEA FILE=BIOSIS ABB=ON  PLU=ON  KALININ# OR LAMININ# OR
                MEROSIN# OR NICEIN#
L46 (      3272)SEA FILE=BIOSIS ABB=ON  PLU=ON  GAMMA2 OR GAMMA 2
L47 (    644158)SEA FILE=BIOSIS ABB=ON  PLU=ON  ANTIBOD? OR IMMUNOGLOB? OR IG#
                OR MAB
L48 (    210831)SEA FILE=BIOSIS ABB=ON  PLU=ON  METASTA? OR INVASI? OR INVAD?
L49 (     17404)SEA FILE=BIOSIS ABB=ON  PLU=ON  L45 OR L46
L50 (   2872541)SEA FILE=BIOSIS ABB=ON  PLU=ON  TREAT? OR INHIBIT? OR THERAP?
L51 (     35650)SEA FILE=BIOSIS ABB=ON  PLU=ON  L44 (5A) L50
L52 (      1055)SEA FILE=BIOSIS ABB=ON  PLU=ON  L49 (3A) L47
L53 (         7)SEA FILE=BIOSIS ABB=ON  PLU=ON  L51 AND L52
L54           1 SEA FILE=BIOSIS ABB=ON  PLU=ON  L53 AND L48


=> S L22 OR L32 OR L54
L166          5 L22 OR L32 OR L54

=> FILE BIOTECHNO
FILE 'BIOTECHNO' ENTERED AT 17:13:19 ON 21 JUN 2002
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FILE LAST UPDATED: 18 JUN 2002       <20020618/UP>
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FILE COVERS 1980 TO DATE.

>>>  SIMULTANEOUS LEFT AND RIGHT TRUNCATION AVAILABLE IN
     /CT AND BASIC INDEX  <<<

=> D QUE L65
L55 (      4086)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  KALININ# OR LAMININ# OR
                 MEROSIN# OR NICEIN#
L56 (      1278)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  GAMMA2 OR GAMMA 2
L57 (    236558)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  ANTIBOD? OR IMMUNOGLOB? OR
                 IG# OR MAB
L58 (     34776)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  METASTA? OR INVASI? OR
                 INVAD?
L59 (    480324)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  TREAT? OR INHIBIT? OR
                 THERAP?
L60 (      5264)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  L55 OR L56
L61 (      4178)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  L58 (5A) L59
L62 (       454)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  L60 (5A) L57
L63 (         8)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  L61 AND L62
L64 (         3)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  L63 AND (ANTILAMININ OR
                 LAMININ-MONOCLONAL OR ANTI-LAMININ) (2W) ANTIBOD?
L65           2 SEA FILE=BIOTECHNO ABB=ON  PLU=ON  L64 NOT TRYPANOSOMA/TI


=> D QUE L79
L66 (     56274)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  ?CARCINOMA?
L67 (      1309)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  (INTRAEPITHELIAL OR INTRA
                 EPITHELIAL) (W) NEOPLAS? OR BOWEN? DISEASE OR BASAL (2A) NEVUS
                 OR LINITIS PLASTICA
L68 (       326)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  (CARCINOID OR KLATSKIN? OR
                 KRUKENBURG?) (1W) (TUMOR OR TUMOUR OR SYNDROME)
L69 (      4086)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  KALININ# OR LAMININ# OR
                 MEROSIN# OR NICEIN#
L70 (      1278)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  GAMMA2 OR GAMMA 2
L71 (    236558)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  ANTIBOD? OR IMMUNOGLOB? OR
                 IG# OR MAB
L72 (     34776)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  METASTA? OR INVASI? OR
                 INVAD?
L73 (    480324)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  TREAT? OR INHIBIT? OR
                 THERAP?
L74 (     56794)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  (L66 OR L67 OR L68)
L75 (      5264)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  L69 OR L70
L76 (       454)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  L75 (5A) L71
L77 (      3957)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  L74 (5A) L73
L78 (         3)SEA FILE=BIOTECHNO ABB=ON  PLU=ON  L77 AND L76
L79           1 SEA FILE=BIOTECHNO ABB=ON  PLU=ON  L78 AND L72


=> S L65 OR L79
L167          3 L65 OR L79

=> FILE EMBASE
FILE 'EMBASE' ENTERED AT 17:13:54 ON 21 JUN 2002
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 FILE COVERS 1974 TO 20 Jun 2002 (20020620/ED)

 EMBASE has been reloaded. Enter HELP RLOAD for details.

 This file contains CAS Registry Numbers for easy and accurate
```

substance identification.

```
=> D QUE L82
L80 (         4) SEA FILE=EMBASE ABB=ON  PLU=ON  LAMININ 5 GAMMA2/CT OR LAMININ
                 GAMMA 2/CT
L81 (    252243) SEA FILE=EMBASE ABB=ON  PLU=ON  ANTIBODY+NT,PFT/CT
L82           0  SEA FILE=EMBASE ABB=ON  PLU=ON  L80 AND L81


=> D QUE L91
L83 (    252243) SEA FILE=EMBASE ABB=ON  PLU=ON  ANTIBODY+NT,PFT/CT
L84 (      7084) SEA FILE=EMBASE ABB=ON  PLU=ON  KALININ+PFT/CT OR LAMININ+PFT/C
                 T
L85 (    252258) SEA FILE=EMBASE ABB=ON  PLU=ON  CARCINOMA+NT,PFT/CT
L86 (    103729) SEA FILE=EMBASE ABB=ON  PLU=ON  METASTASIS+NT,PFT/CT OR CANCER
                 INVASION+PFT/CT
L87 (     11135) SEA FILE=EMBASE ABB=ON  PLU=ON  L86 (L) (DT OR PC OR TH)/CT
L88 (     25668) SEA FILE=EMBASE ABB=ON  PLU=ON  L83 (L) (AD OR DO OR DT OR PK
                 OR PD)/CT
L89 (        31) SEA FILE=EMBASE ABB=ON  PLU=ON  L84 AND L88
L90 (         2) SEA FILE=EMBASE ABB=ON  PLU=ON  L89 AND L87
L91           0  SEA FILE=EMBASE ABB=ON  PLU=ON  L90 AND L85


=> D QUE L98
L92 (    252243) SEA FILE=EMBASE ABB=ON  PLU=ON  ANTIBODY+NT,PFT/CT
L93 (      7084) SEA FILE=EMBASE ABB=ON  PLU=ON  KALININ+PFT/CT OR LAMININ+PFT/C
                 T
L94 (    252258) SEA FILE=EMBASE ABB=ON  PLU=ON  CARCINOMA+NT,PFT/CT
L95 (     25668) SEA FILE=EMBASE ABB=ON  PLU=ON  L92 (L) (AD OR DO OR DT OR PK
                 OR PD)/CT
L96 (        31) SEA FILE=EMBASE ABB=ON  PLU=ON  L93 AND L95
L97 (     30509) SEA FILE=EMBASE ABB=ON  PLU=ON  L94 (L) (DT OR PC OR TH)/CT
L98           0  SEA FILE=EMBASE ABB=ON  PLU=ON  L96 AND L97


=> D QUE L106
L99 (    582312) SEA FILE=EMBASE ABB=ON  PLU=ON  ANTIBOD? OR IMMUNOGLOB? OR IG#
                 OR MAB
L100(    252483) SEA FILE=EMBASE ABB=ON  PLU=ON  CARCINOMA+NT,PFT/CT
L101(     30559) SEA FILE=EMBASE ABB=ON  PLU=ON  L100 (L) (DT OR PC OR TH)/CT
L102(     10347) SEA FILE=EMBASE ABB=ON  PLU=ON  KALININ# OR LAMININ# OR
                 MEROSIN# OR NICEIN#
L103(      2861) SEA FILE=EMBASE ABB=ON  PLU=ON  GAMMA2 OR GAMMA 2
L104(     13061) SEA FILE=EMBASE ABB=ON  PLU=ON  L102 OR L103
L105(       918) SEA FILE=EMBASE ABB=ON  PLU=ON  L104 (3A) L99
L106          0  SEA FILE=EMBASE ABB=ON  PLU=ON  L101 AND L105


=> D QUE L114
L107(    582312) SEA FILE=EMBASE ABB=ON  PLU=ON  ANTIBOD? OR IMMUNOGLOB? OR IG#
                 OR MAB
L108(    103868) SEA FILE=EMBASE ABB=ON  PLU=ON  METASTASIS+NT,PFT/CT OR CANCER
                 INVASION+PFT/CT
L109(     11152) SEA FILE=EMBASE ABB=ON  PLU=ON  L108 (L) (DT OR PC OR TH)/CT
L110(     10347) SEA FILE=EMBASE ABB=ON  PLU=ON  KALININ# OR LAMININ# OR
                 MEROSIN# OR NICEIN#
L111(      2861) SEA FILE=EMBASE ABB=ON  PLU=ON  GAMMA2 OR GAMMA 2
L112(     13061) SEA FILE=EMBASE ABB=ON  PLU=ON  L110 OR L111
L113(       918) SEA FILE=EMBASE ABB=ON  PLU=ON  L112 (3A) L107
```

DT = Drug Therapy
PC = Prevention and control
TH = Therapy
AD = Administration
DO = Drug Dose
PK = pharmacokinetics
PD = Pharmacology

L114 2 SEA FILE=EMBASE ABB=ON PLU=ON L109 AND L113

=> FILE HCAPLUS
FILE 'HCAPLUS' ENTERED AT 17:15:02 ON 21 JUN 2002
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

FILE COVERS 1907 - 21 Jun 2002 VOL 136 ISS 25
FILE LAST UPDATED: 19 Jun 2002 (20020619/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

CAS roles have been modified effective December 16, 2001. Please check your SDI profiles to see if they need to be revised. For information on CAS roles, enter HELP ROLES at an arrow prompt or use the CAS Roles thesaurus (/RL field) in this file.

=> D QUE L119
L115(373) SEA FILE=HCAPLUS ABB=ON PLU=ON "CARCINOMA (L) METASTASIS"+PFT /CT
L116(6434) SEA FILE=HCAPLUS ABB=ON PLU=ON LAMININS+NT,PFT/CT
L117(195305) SEA FILE=HCAPLUS ABB=ON PLU=ON ANTIBODIES+NT,PFT/CT
L118(31724) SEA FILE=HCAPLUS ABB=ON PLU=ON L117 (L) (THU OR BAC)/RL
L119 1 SEA FILE=HCAPLUS ABB=ON PLU=ON L115 AND L116 AND L118

=> D QUE L127
L120(6434) SEA FILE=HCAPLUS ABB=ON PLU=ON LAMININS+NT,PFT/CT
L121(195305) SEA FILE=HCAPLUS ABB=ON PLU=ON ANTIBODIES+NT,PFT/CT
L122(31724) SEA FILE=HCAPLUS ABB=ON PLU=ON L121 (L) (THU OR BAC)/RL
L123(18169) SEA FILE=HCAPLUS ABB=ON PLU=ON CARCINOMA+NT,PFT/CT
L124(140647) SEA FILE=HCAPLUS ABB=ON PLU=ON ANTITUMOR AGENTS+NT,PFT/CT
L125(175) SEA FILE=HCAPLUS ABB=ON PLU=ON L123 AND L120
L126(7) SEA FILE=HCAPLUS ABB=ON PLU=ON L125 AND L124
L127 3 SEA FILE=HCAPLUS ABB=ON PLU=ON L126 AND L122

=> D QUE L136
L128(373) SEA FILE=HCAPLUS ABB=ON PLU=ON "CARCINOMA (L) METASTASIS"+PFT /CT
L129(18169) SEA FILE=HCAPLUS ABB=ON PLU=ON CARCINOMA+NT,PFT/CT
L130(9355) SEA FILE=HCAPLUS ABB=ON PLU=ON KALININ# OR LAMININ# OR MEROSIN# OR NICEIN#
L131(10738) SEA FILE=HCAPLUS ABB=ON PLU=ON GAMMA2 OR GAMMA 2
L132(438603) SEA FILE=HCAPLUS ABB=ON PLU=ON ANTIBOD? OR IMMUNOGLOB? OR IG# OR MAB

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L133(     19940)SEA FILE=HCAPLUS ABB=ON  PLU=ON  L130 OR L131
L134(       987)SEA FILE=HCAPLUS ABB=ON  PLU=ON  L133 (3A) L132
L135(        19)SEA FILE=HCAPLUS ABB=ON  PLU=ON  L129 AND L134
L136          2 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L135 AND L128


=> S L119 OR L127 OR L136
L168          5 L119 OR L127 OR L136

=> FILE WPIDS
FILE 'WPIDS' ENTERED AT 17:16:00 ON 21 JUN 2002
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FILE LAST UPDATED: 18 JUN 2002              <20020618/UP>
MOST RECENT DERWENT UPDATE            200238   <200238/DW>
DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE

>>> The BATCH option for structure searches has been
    enabled in WPINDEX/WPIDS and WPIX  >>>

>>> PATENT IMAGES AVAILABLE FOR PRINT AND DISPLAY >>>

>>> FOR DETAILS OF THE PATENTS COVERED IN CURRENT UPDATES,
    SEE http://www.derwent.com/dwpi/updates/dwpicov/index.html <<<

>>> FOR A COPY OF THE DERWENT WORLD PATENTS INDEX TOOLS OF THE
    TRADE USER GUIDE, PLEASE VISIT:
    http://www.derwent.com/data/stn3.pdf <<<

>>> FOR INFORMATION ON ALL DERWENT WORLD PATENTS INDEX USER
    GUIDES, PLEASE VISIT:
    http://www.derwent.com/userguides/dwpi_guide.html <<<

=> D QUE L149
L137(      7014)SEA FILE=WPIDS ABB=ON  PLU=ON  ?CARCINOMA?
L138(        83)SEA FILE=WPIDS ABB=ON  PLU=ON  (INTRAEPITHELIAL OR INTRA
                  EPITHELIAL) (W) NEOPLAS? OR BOWEN? DISEASE OR BASAL (2A) NEVUS
                  OR LINITIS PLASTICA
L139(       106)SEA FILE=WPIDS ABB=ON  PLU=ON  (CARCINOID OR KLATSKIN? OR
                  KRUKENBURG?) (1W) (TUMOR OR TUMOUR OR SYNDROME)
L140(       428)SEA FILE=WPIDS ABB=ON  PLU=ON  KALININ# OR LAMININ# OR
                  MEROSIN# OR NICEIN#
L141(       525)SEA FILE=WPIDS ABB=ON  PLU=ON  GAMMA2 OR GAMMA 2
L142(     50245)SEA FILE=WPIDS ABB=ON  PLU=ON  ANTIBOD? OR IMMUNOGLOB? OR IG#
                  OR MAB
L143(     18328)SEA FILE=WPIDS ABB=ON  PLU=ON  METASTA? OR INVASI? OR INVAD?
L144(      7137)SEA FILE=WPIDS ABB=ON  PLU=ON  (L137 OR L138 OR L139)
L145(       947)SEA FILE=WPIDS ABB=ON  PLU=ON  L140 OR L141
L146(        31)SEA FILE=WPIDS ABB=ON  PLU=ON  L144 AND L145
L147(        18)SEA FILE=WPIDS ABB=ON  PLU=ON  L146 AND L142
L148(         7)SEA FILE=WPIDS ABB=ON  PLU=ON  L147 AND L143
L149          4 SEA FILE=WPIDS ABB=ON  PLU=ON  L148 AND (TREAT?/TI OR INHIBIT?/
                  TI OR THERAP?/TI)


=> D QUE L154
L150(       428)SEA FILE=WPIDS ABB=ON  PLU=ON  KALININ# OR LAMININ# OR
                  MEROSIN# OR NICEIN#
L151(       525)SEA FILE=WPIDS ABB=ON  PLU=ON  GAMMA2 OR GAMMA 2
L152(     18328)SEA FILE=WPIDS ABB=ON  PLU=ON  METASTA? OR INVASI? OR INVAD?
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L153(          6)SEA FILE=WPIDS ABB=ON  PLU=ON  L150 AND L151
L154           1 SEA FILE=WPIDS ABB=ON  PLU=ON  L153 AND L152


=> D QUE L164
L155(       7014)SEA FILE=WPIDS ABB=ON  PLU=ON  ?CARCINOMA?
L156(         83)SEA FILE=WPIDS ABB=ON  PLU=ON  (INTRAEPITHELIAL OR INTRA
                    EPITHELIAL) (W) NEOPLAS? OR BOWEN? DISEASE OR BASAL (2A) NEVUS
                    OR LINITIS PLASTICA
L157(        106)SEA FILE=WPIDS ABB=ON  PLU=ON  (CARCINOID OR KLATSKIN? OR
                    KRUKENBURG?) (1W) (TUMOR OR TUMOUR OR SYNDROME)
L158(        428)SEA FILE=WPIDS ABB=ON  PLU=ON  KALININ# OR LAMININ# OR
                    MEROSIN# OR NICEIN#
L159(        525)SEA FILE=WPIDS ABB=ON  PLU=ON  GAMMA2 OR GAMMA 2
L160(      50245)SEA FILE=WPIDS ABB=ON  PLU=ON  ANTIBOD? OR IMMUNOGLOB? OR IG#
                    OR MAB
L161(       7137)SEA FILE=WPIDS ABB=ON  PLU=ON  (L155 OR L156 OR L157)
L162(        947)SEA FILE=WPIDS ABB=ON  PLU=ON  L158 OR L159
L163(         30)SEA FILE=WPIDS ABB=ON  PLU=ON  L162 (3A) L160
L164           3 SEA FILE=WPIDS ABB=ON  PLU=ON  L163 AND L161


=> S L149 OR L154 OR L164
L169           7 L149 OR L154 OR L164

=> FIL STNGUIDE
FILE 'STNGUIDE' ENTERED AT 17:17:29 ON 21 JUN 2002
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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Jun 14, 2002 (20020614/UP).

=> DUP REM L165 L166 L167 L114 L168 L169
FILE 'CANCERLIT' ENTERED AT 17:20:29 ON 21 JUN 2002

FILE 'BIOSIS' ENTERED AT 17:20:29 ON 21 JUN 2002
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PROCESSING COMPLETED FOR L165
PROCESSING COMPLETED FOR L166
PROCESSING COMPLETED FOR L167
PROCESSING COMPLETED FOR L114
PROCESSING COMPLETED FOR L168
PROCESSING COMPLETED FOR L169
L170          26 DUP REM L165 L166 L167 L114 L168 L169 (4 DUPLICATES REMOVED)
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=> D IBIB AB CT 1-26

L170 ANSWER 1 OF 26 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 2002-089823 [12] WPIDS
DOC. NO. CPI: C2002-027679
TITLE: Regulating laminin 5 expression or activity for treating squamous cell **carcinoma**, glioma, by contacting laminin 5 with an agent that affects processing of laminin 5 by a bone morphogenetic protein-1 related protein.
DERWENT CLASS: B04 D16
INVENTOR(S): FINDELL, P R; MARINKOVICH, M P
PATENT ASSIGNEE(S): (FIBR-N) FIBROGEN INC; (STRD) UNIV LELAND STANFORD JUNIOR
COUNTRY COUNT: 96
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001087239	A2	20011122	(200212)*	EN	102

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

AU 2001061519 A 20011126 (200222)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001087239	A2	WO 2001-US15417	20010511
AU 2001061519	A	AU 2001-61519	20010511

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001061519	A Based on	WO 200187239

PRIORITY APPLN. INFO: US 2000-203708P 20000512
AB WO 200187239 A UPAB: 20020221
NOVELTY - Regulating (M1) laminin 5 expression or activity, comprising contacting laminin 5 with an agent (A) that affects processing of laminin 5 by a bone morphogenetic protein-1 (BMP-1) related protein, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:
(1) a composition for treating a condition associated with increased expression or activity of laminin 5, comprising (A);
(2) diagnosing (M2) the presence of a condition characterized by increased expression or activity of laminin 5 in a subject, comprising:
(a) obtaining a sample;
(b) detecting the level of expression or activity of a BMP-1 related protein in the sample; and
(c) comparing the level of expression or activity of the BMP-1 related protein in the sample to a standard level of expression or activity of the BMP-1 related protein;
(3) a diagnostic kit for use in diagnosing the presence of a condition associated with increased expression or activity of laminin 5 in

a sample from a subject, comprising an anti-BMP-1 antibody reactive with BMP-1 related proteins and a labeled reagent capable of forming a complex with a BMP-1 related protein or with the anti-BMP-1 antibody

(4) screening for an agent that affects the processing of laminin 5 by BMP-1 related proteins, by contacting a sample containing unprocessed laminin 5 with BMP related protein and the agent, measuring and comparing the level of processed laminin 5 in the sample to a control sample;

(5) an isolated polypeptide (I) comprising a BMP-1 cleavage sequence (S2);

(6) an isolated polypeptide comprising a BMP-1 cleavage sequence chosen from (S1);

(7) an isolated polynucleotide encoding (I);

(8) an isolated polynucleotide that is complement to the polynucleotide of (7); and

(9) an antibody (II) that binds to (I).

S1 is LeuGlnPheGlyAspIleProThr, GlnLeuLeuGlnAspThrProValAla, LysValTrpGlnAspAlaCysSer and GlnPheAlaValAspMetGlnThr. S2 is CysTyrSerGlyAspGluAsnPro.

ACTIVITY - Cytostatic.

MECHANISM OF ACTION - Inhibitor of processing of laminin 5 by a BMP-1 related protein.

The processing of laminin 5 by various BMP-1 related proteins was examined. Unprocessed laminin 5 was deposited onto the surface of culture dishes by keratinocytes cultured in the presence of 10 micro M inhibitor of BMP-1 activity, which prevented processing of laminin 5. The cells were removed from the culture dish by 20 mM ammonium sulfate, the dish was washed, and the matrix was incubated in increasing amounts of each BMP-1 related protein. After proteolytic digestion, the matrix was extracted and examined by Western blot analysis. The results showed that BMP-1, mTld, mTll-1, and mTll-2 processed laminin 5. BMP-1, mTld, mTll-1 and mTll-2 all cleaved the alpha 3 chain of laminin 5. BMP-1 and mTll-2 cleaved the gamma 2 chain of laminin 5. Additionally, mTll-2 showed more potent cleavage activity towards the alpha 3 chain and gamma 2 chain of laminin 5 compared to the other BMP-1 related proteins. The inhibitor at 10 micro M inhibited cleavage of laminin 5 gamma 2 chain by BMP-1 and mTll-2, and inhibited cleavage of laminin 5 alpha 3 chain by BMP-1, mTll-1, and mTll-2.

USE - (M1) is useful for affecting laminin 5 expression or activity, for treating a condition characterized by increased expression or activity of laminin 5, especially cancer, glioma, a condition characterized by neoplastic epithelial cells chosen from squamous cells, keratinocytes, mucosal epithelial cells, gastrointestinal epithelial cells, corneal epithelia of the eye and epithelial cells of the urinary and reproductive tract, squamous cell **carcinoma** such as cancers of the skin, lung, head, neck, oral, cervical, tongue, gastric, colorectal, throat, cancer of the urinary tract, reproductive tract, esophageal cancer and bronchiogenic **carcinoma**. (M2) is useful for diagnosing the presence of squamous cell **carcinoma** in a subject by detecting the level of expression of BMP-1 related protein in tissue, urine, serum or blood sample. (I) is useful for screening for an agent that affects the processing of laminin 5 by BMP-1 related proteins by contacting sample containing the polypeptide with a BMP-1 related protein and an agent, measuring and comparing the level of the polypeptide that is processed in the sample to a control sample. (All claimed). (II) is useful to identify BMP-1 related proteins, laminin 5 or processed laminin 5 or laminin 5 chains, or their fragments or subunits, in a sample e.g. from biopsied tissue, and to inhibit processing of laminin 5 by BMP-1 related proteins.

Dwg.0/26

L170 ANSWER 2 OF 26 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 2001-514501 [56] WPIDS

DOC. NO. CPI: C2001-153732
TITLE: Composition comprising a combination of an oxidizing and/or reducing agent, a protein-denaturing agent, and a hapten, useful for **treating** neoplasms, tumors, and cancers.
DERWENT CLASS: B05 D16
INVENTOR(S): YU, B
PATENT ASSIGNEE(S): (YUBB-I) YU B
COUNTRY COUNT: 94
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001052868	A1	20010726	(200156)*	EN	83

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW

AU 2001030977	A	20010731	(200171)		
US 2002044919	A1	20020418	(200228)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001052868	A1	WO 2001-US1737	20010118
AU 2001030977	A	AU 2001-30977	20010118
US 2002044919	A1 Provisional	US 2000-177024P	20000119
		US 2001-765060	20010117

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001030977	A Based on	WO 200152868

PRIORITY APPLN. INFO: US 2000-177024P 20000119; US 2001-765060 20010117

AB WO 200152868 A UPAB: 20011001
NOVELTY - A composition (I) comprising a combination of an oxidizing or reducing agent, a protein-denaturing agent, and a hapten, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a kit comprising the combination (I);
(2) an article of manufacture comprising:
(a) packaging material;
(b) the combination above; and
(c) a label indicating that the article is for treating neoplasms; and

(3) a method for treating neoplasm in a mammal comprising in situ administration to the neoplasm of a mammal, a hapten and a coagulation agent or treatment that causes coagulation of the neoplasm (an autologous immune response is generated against the neoplasm).

ACTIVITY - Cytostatic.

31 advanced stage IV liver cancer patients were treated using the new combination. Prior to procedure, patients were given a mild sedative or painkiller. Patients were calmed thoroughly and were also monitored by modern medial imaging. With local anesthesia, percutaneous puncture was

administered directly into the tumor using a spinal needle connected to a high-power syringe containing a combination of ethanol, H2O2, anticancer drug AraC (8 mg/ml) and hemotoxilin (5 mg/ml). Combination was injected directly into the tumor and distributed throughout the matrix of the whole tumor. Sonic imaging showed the stranger echo imaging which indicated the coagulation area.

Following coagulation lysis and tumor cell death monitored by sonic imaging, which showed liquefied echo, tumor started to shrink and disappear. Normal tissues grew replacing the tumor. The process was monitored by medical imaging systems. The amount of the ingredients of the combination injected into the tumor was determined by the diameter of tumors (cm) with 2 ml of the combination for each centimeter.

Procedure was repeated in 1-2 weeks. On average, each patient was treated with the injection for 3 times. No severe side effects for all the treated patients was observed, although some patients experienced tolerable pain the injection site while a few had light fever during the first week. All side effects disappeared in about 1 week. No serious complications happened in any cases.

MECHANISM OF ACTION - Gene therapy.

USE - The combination and the methods are useful for treating neoplasms, tumors, and cancers, including neoplasm or cancer of the e.g. adrenal gland, anus, auditory nerve, bile ducts, bladder, bone, brain, breast, bruccal, central nervous system, cervix, colon, ear, endometrium, esophagus, eye, eyelids, fallopian tube, gastrointestinal tract, head and neck, heart, kidney, larynx, liver, lung, or mandible.

The combination and methods may further be used in treating tumors of mesenchymal origin (e.g. connective tissue and derivatives, or endothelial and related tissues blood vessels), epithelial origin (stratified squamous **carcinoma**, or basal cells of skin or adenexa), and tumors derived from more than one neoplastic cell types derived from more than one germ layers.

The treatment may be used with radiation therapy, before surgery for the pre-treatment of neoplasm for easier removal of the neoplastic mass and reduces the neoplasm **metastasis** rate, or with gene therapy.

Dwg.0/4

L170 ANSWER 3 OF 26 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:53712 HCAPLUS
DOCUMENT NUMBER: 132:106963
TITLE: Compounds and methods for modulating cadherin-mediated functions
INVENTOR(S): Doherty, Patrick; Blaschuk, Orest W.; Gour, Barbara J.
PATENT ASSIGNEE(S): Adherex Technologies, Inc., Can.
SOURCE: PCT Int. Appl., 144 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000002917	A2	20000120	WO 1999-CA627	19990712
WO 2000002917	A3	20000504		

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 6277824 B1 20010821 US 1998-113977 19980710
AU 9945964 A1 20000201 AU 1999-45964 19990712
EP 1097168 A2 20010509 EP 1999-928963 19990712
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO

PRIORITY APPLN. INFO.: US 1998-113977 A 19980710
WO 1999-CA627 W 19990712

AB Modulating agents and methods for enhancing or inhibiting cadherin-mediated functions are provided. The modulating agents comprise at least an HAV binding motif, an analog or peptidomimetic thereof, or an antibody or fragment thereof that specifically binds to such a motif. Modulating agents may addnl. comprise one or more cell adhesion recognition sequences recognized by cadherins and/or other adhesion mols. Such modulating agents may, but need not, be linked to a targeting agent, drug and/or support material.

CT Cadherins
CT Polynucleotides
CT Protein motifs
CT Acetyl group
CT Cadherins
CT Cell adhesion molecules
CT Cell migration
CT Transplant and Transplantation
CT Gingiva
CT Skin
CT Thioethers
CT Animal cell
CT Drug delivery systems
CT Neoplasm
CT Nervous system
CT Nervous system
CT Chemistry
CT Cell adhesion molecules
CT Amide group
CT Angiogenesis
CT Bioreactors
CT **Carcinoma**
CT Cell adhesion
CT Disulfide group
CT Drug delivery systems
CT Drug screening
CT Drug targeting
CT Drugs
CT Epithelium
CT Fluorescent substances
CT Immunostimulation
CT Immunosuppression
CT Labels
CT Leukemia
CT Lymphocyte
CT Melanoma
CT Membranes, nonbiological
CT Microparticles
CT Multiple sclerosis
CT Oligodendrocyte
CT Ovary, neoplasm
CT Peptidomimetics

CT Protein sequences
CT Test kits
CT Ultrathin films
CT Wound healing
CT Cadherins
CT Fibronectins
CT Integrins
CT **Laminins**
CT Antigens
CT **Antibodies**
CT Plastics, biological studies
CT Peptides, biological studies
CT Glycoproteins, specific or class
CT Glycoproteins, specific or class
CT **Antitumor agents**
CT Blood vessel
CT Proteins, specific or class
CT Apoptosis
CT Spinal cord
CT Cell adhesion molecules
CT Polymers, biological studies
CT Neoplasm
CT Schwann cell
CT Bladder
CT Nerve
CT Cell adhesion molecules
CT Axon
CT Blood vessel
CT Blood vessel
CT Biological transport
CT Medical goods
CT Pregnancy
CT Oligodendrocyte
CT Blood
CT Drug delivery systems
CT Transplant and Transplantation
CT Transplant and Transplantation
CT Synapse
CT Drug delivery systems
CT Medical goods
CT Drug delivery systems
CT Skin
CT Skin
CT Medical goods

L170 ANSWER 4 OF 26 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 2000-679729 [66] WPIDS
DOC. NO. CPI: C2000-206817
TITLE: Gene-transfer vector containing an enhancer-promoter, useful for prevention or **treatment** of tumors, is able to enter normal tissue to prevent **metastasis**.
DERWENT CLASS: B04 D16
INVENTOR(S): BAKER, A; BRAND, K; STRAUSS, M
PATENT ASSIGNEE(S): (BRAN-I) BRAND K
COUNTRY COUNT: 93
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG

WO 2000066176 A2 20001109 (200066)* GE 23
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
DE 10022687 A1 20010201 (200108)
AU 2000058022 A 20001117 (200111)
EP 1173225 A2 20020123 (200214) GE
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000066176	A2	WO 2000-DE1416	20000502
DE 10022687	A1	DE 2000-10022687	20000502
AU 2000058022	A	AU 2000-58022	20000502
EP 1173225	A2	EP 2000-943552	20000502
		WO 2000-DE1416	20000502

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000058022	A	Based on	WO 200066176
EP 1173225	A2	Based on	WO 200066176

PRIORITY APPLN. INFO: DE 1999-19919865 19990430

AB WO 200066176 A UPAB: 20001219

NOVELTY - An agent (A) for prophylaxis and treatment of tumors comprising a vector (I), an enhancer-promoter (II) and a transgene (III), with at least 1 of these components being able to enter normal cells, is new.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a similar agent (A') containing an antitumor transgene (IIIa), or its sequences, provided with a membrane anchor sequence.

ACTIVITY - Cytostatic.

MECHANISM OF ACTION - Inhibition of enzymes that break down extracellular matrix, increasing collagen content of tissue, or improving cell-cell and cell-matrix adhesion, all of which lead to reduced **metastatic invasion**.

Liver **metastases** were induced in mice by injecting 2 million LS174 cells into the spleen, 3 days after administration of 3 multiply 1010 plaque-forming units of an adenoviral vector expressing the TIMP-2 metalloproteinase inhibitor. After 4 weeks, the mean weight of liver tumors was 200 mg, compared with 3200 mg in untreated controls.

USE - (A) are especially used for treatment and prevention of liver **metastases** or for treating brain tumors and lung **metastases**. Typically they are used pre- or intra-operatively, to suppress spread of tumor cells released from primary tumors during surgery, for long-term suppression, or killing, of occult micrometastases, especially in high-risk groups such as patients who have undergone surgery for breast cancer with lymph node involvement or to limit growth of established **metastases** or inoperable primary tumors (e.g. glioblastoma or hepatocellular **carcinoma**).

ADVANTAGE - (A) are able to enter normal tissue so emergence and/or further growth of **metastases** is prevented, and thus further spread of inoperable primary cancers. Gene therapy allows very high

concentrations of active agents to be generated at target sites, with reduced systemic side effects, several different active agents can be administered simultaneously, and a single administration provides long-lasting gene expression.
Dwg.0/2

L170 ANSWER 5 OF 26 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:233936 HCAPLUS
DOCUMENT NUMBER: 130:266373
TITLE: Compounds and methods for regulating cell adhesion
INVENTOR(S): Blaschuck, Orest W.; Gour, Barbara J.
PATENT ASSIGNEE(S): Adherex Inc., Can.
SOURCE: PCT Int. Appl., 148 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9916791	A2	19990408	WO 1998-CA902	19980929
WO 9916791	A3	19990520		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6203788	B1	20010320	US 1997-939853	19970929
AU 9892483	A1	19990423	AU 1998-92483	19980929

PRIORITY APPLN. INFO.: US 1997-939853 A 19970929
WO 1998-CA902 W 19980929

AB Methods for using modulating agents to enhance or inhibit cadherin-mediated cell adhesion in a variety of in vivo and in vitro contexts are provided. In particular, the modulating agents may be used in the therapy of multiple sclerosis and other demyelinating diseases. The modulating agents comprise at least one cadherin cell adhesion recognition sequence (HAV) or an antibody or fragment thereof that specifically binds to a cadherin cell adhesion recognition sequence. Modulating agents may addnl. comprise one or more cell adhesion recognition sequences recognized by other adhesion mols. Such modulating agents may, but need not, be linked to a targeting agent, drug and/or support material.

CT Cadherins
CT Cell adhesion molecules
CT Cell migration
CT Astrocyte
CT **Angiogenesis inhibitors**
CT Animal tissue
CT Apoptosis
CT **Carcinoma**
CT Circulation
CT Disease, animal
CT Drugs
CT Epithelium
CT Fluorescent substances
CT Immunomodulators

CT Inflammation
CT Injury
CT Leukemia
CT Mammal (Mammalia)
CT Melanoma
CT Microfilament
CT Multiple sclerosis
CT Neoplasm
CT Oligodendrocyte
CT Ovary, neoplasm
CT Protein sequences
CT Schwann cell
CT Skin
CT Surgery
CT Test kits
CT Transplant and Transplantation
CT Wound healing
CT Cadherins
CT **Antibodies**
CT Animal cell
CT Drug delivery systems
CT Nervous system
CT Nerve, disease
CT Nerve, disease
CT Blood vessel
CT Drug delivery systems
CT Prosthetic materials and Prosthetics
CT Spinal cord
CT Rat
CT Cell adhesion
CT Bladder
CT Nerve
CT Axon
CT Blood vessel
CT Biological transport
CT Pregnancy
CT Oligodendrocyte
CT Kidney
CT Drug delivery systems
CT Synapse
CT **Laminins**
CT Drug delivery systems

L170 ANSWER 6 OF 26 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 1999161283 EMBASE
TITLE: Inhibition of experimental metastasis of human fibrosarcoma cells by anti-recombinant 37-kDa **laminin** binding protein **antibody**.
AUTHOR: Narumi K.; Inoue A.; Tanaka M.; Isemura M.; Shimo-Oka T.; Abe T.; Nukiwa T.; Satoh K.
CORPORATE SOURCE: K. Satoh, Dept. Respir. Oncology Mol. Medicine, Inst. Development Aging and Cancer, Tohoku University, 4-1 Seiryo-machi, Aoba-ku, Sendai 980-8575, Japan. kensatoh@idac.tohoku.ac.jp
SOURCE: Japanese Journal of Cancer Research, (1999) 90/4 (425-431). Refs: 33
ISSN: 0910-5050 CODEN: JJCREP
COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 013 Dermatology and Venereology

016 Cancer
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The laminin binding protein of 37 kDa (37LBP) is regarded as a precursor protein of the high-affinity 67-kDa laminin receptor (67LR). Expression of 67LR/37LBP is well correlated with biological aggressiveness of cancer, particularly with invasive and metastatic potential. To investigate in detail the role of 37LBP in cancer cells, we synthesized recombinant 37LBP (r37LBP) as a fusion protein and generated an IgG-type polyclonal antibody P4G against r37LB. Western blot analysis with P4G showed a single band of 67LR under both nonreducing and reducing conditions using cell extract of human fibrosarcoma cells HT1080. It was shown that P4G inhibited cell attachment to immobilized laminin in a dose-dependent manner. Further, the intravenous injection of NT1080 cells pretreated with P4G, compared with that of cells pretreated with normal rabbit serum, resulted in a reduced number of experimental metastases (3.3 .+-. 5.1 vs. 58.0 .+-. 38.0 nodules per mouse, respectively) ($P < 0.005$). These results suggest that P4G inhibits the colonization and growth of HT1080 cells in the lungs of mice, and that the blocking of r37LBP with the specific antibody P4G may offer a potential strategy for preventing cancer metastasis.
CT Medical Descriptors:
*fibrosarcoma
***metastasis: PC, prevention**
cancer inhibition
immunoblotting
rabbit
serum
lung
human
nonhuman
mouse
animal experiment
animal model
controlled study
human cell
animal tissue
article
priority journal
Drug Descriptors:
*laminin binding protein
*protein antibody
recombinant protein
protein precursor
polyclonal antibody
immunoglobulin g antibody
hybrid protein
laminin receptor: EC, endogenous compound

L170 ANSWER 7 OF 26 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 1998-240534 [21] WPIDS
DOC. NO. NON-CPI: N1998-190279
DOC. NO. CPI: C1998-075107
TITLE: Use of laminin and fragments - for developing products for use in the diagnosis and treatment of amyloid disease, e.g. Alzheimer's disease or CJD.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): CASTILLO, G; SNOW, A D
PATENT ASSIGNEE(S): (UNIW) UNIV WASHINGTON
COUNTRY COUNT: 78
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9815179	A1	19980416	(199821)*	EN	141
RW: AT BE CH DE DK EA ES FI FR GB GH GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG UZ VN YU ZW					
AU 9747492	A	19980505	(199836)		
EP 959682	A1	19991201	(200001)	EN	
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9815179	A1	WO 1997-US18145	19971008
AU 9747492	A	AU 1997-47492	19971008
EP 959682	A1	EP 1997-910016	19971008
		WO 1997-US18145	19971008

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9747492	A	Based on	WO 9815179
EP 959682	A1	Based on	WO 9815179

PRIORITY APPLN. INFO: US 1996-27981P 19961008

AB WO 9815179 A UPAB: 19980528

A method for treating an amyloid disease in a patient is claimed, comprising administering a polypeptide having a conformational similarity to a fragment of a laminin protein.

Also claimed are: (1) a method for the treatment of a patient having an identified clinical need to interfere with the pathological effects of amyloid, comprising administering to the patient a polypeptide selected from a human laminin, or a mouse laminin (11, 177, 177, 3084, 3075, 1786,1786, 1801, 1798, 1607, 1609 aa sequences given in the specification) or their fragments; (2) a method for diagnosis of a disease or disease susceptibility comprises determining levels of laminin, or a particular laminin-derived protein fragment, in a sample, the levels are indicative of the presence of a disease, susceptibility to a disease, or progression of the disease; (3) a method of making an antibody to a peptide sequence within the 130 kD A- beta -laminin binding fragment present in human biological fluids; (4) a process for diagnosing a disease or a susceptibility to a disease related to an underexpression or overexpression of a polypeptide (11, 177, 177, 3084, 3075, 1786,1786, 1801, 1798, 1607, 1609 aa sequences given in the specification), comprises determining a mutation in a nucleic acid sequence encoding one of the polypeptides or their fragments; (4) a method for detection and quantification of laminin and laminin-derived fragments in biological fluids comprising: (a) allowing a first laminin or **laminin** -derived fragment **antibody** to bind to microtitre wells; (b) adding a quantity of biological fluid to the microtitre wells; (c) incubating the biological fluid to allow binding of any laminin or laminin-derived fragment in the biological fluid to the first antibody on the microtitre wells; (d) adding a second labelled antibody to the microtitre wells where the second labelled **antibody** is against

the **laminin** or laminin-derived fragment, but which is against a different epitope than the first antibody, and allowing the second antibody to bind to any laminin or laminin-derived fragment captured by the first antibody; and (e) detecting bound materials using an appropriate substrate or label; (5) a purified laminin polypeptide fragment that is capable of binding to A- beta amyloid protein where the laminin polypeptide fragment has an A- beta binding site within a globular repeating domain of laminin A chain; (6) a method of in vivo inhibition of A- beta amyloidosis comprising: (a) introducing a vector comprising a DNA sequence encoding a laminin polypeptide fragment that is capable of binding to A-beta amyloid protein, where the laminin polypeptide fragment has an A-beta binding site within a globular repeating domain of laminin A chain; (b) producing the laminin polypeptide fragment in vivo to inhibit A-beta amyloidosis; (7) a method of in vivo inhibition of A-beta as in (6) where the polypeptide is a fourth globular repeat within the human lamminin A chain (177 aa sequence given in the specification).

USE - The products and methods can be used for the diagnosis, prognosis, monitoring and treatment of amyloidoses such as Alzheimer's disease, Down's syndrome and hereditary cerebral haemorrhage with amyloidosis of the Dutch type (where the specific amyloid is known as the beta -amyloid protein of A- beta), the amyloidosis associated with chronic inflammation, various forms of malignancy and Familial Mediterranean Fever (AA amyloid or inflammation-association amyloidosis), the amyloidosis associated with multiple myeloma and other B-cell abnormalities (AL amyloid), the amyloidosis associated with type II diabetes (amylin or islet amyloid), the amyloidosis associated with the prion diseases including Creutzfeldt-Jacob disease, Gertstmann-Straussler syndrome, kuru and animal scrapie (PrP amyloid), the amyloidosis associated with long-term heamodialysis and carpal tunnel syndrome (beta 2-microglobulin amyloid), the amyloidosis associated with senile cardiac amyloid and Familial Amyloidotic Polyneuropathy (prealbumin or transthyretin amyloid), and the amyloidosis associated with endocrine tumours such as medullary **carcinoma** of the thyroid (variant of procalcitonin).

Dwg.0/14

L170 ANSWER 8 OF 26 CANCERLIT
ACCESSION NUMBER: 1999197670 CANCERLIT
DOCUMENT NUMBER: 99197670
TITLE: Differential apoptotic susceptibility to anti-Fas IgM and anticancer drugs in a human endometrial adenocarcinoma cell line HHUA on laminin and type I collagen.
AUTHOR: Chang L; Tanaka T; Umesaki N; Ogita S
CORPORATE SOURCE: Department of Obstetrics & Gynecology, Osaka City University, Medical School, Japan.
SOURCE: OSAKA CITY MEDICAL JOURNAL, (1998). Vol. 44, No. 2, pp. 173-80.
Journal code: OLI. ISSN: 0030-6096.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L
LANGUAGE: English
OTHER SOURCE: MEDLINE 99197670
ENTRY MONTH: 199905

AB Apoptotic susceptibility of epithelial cells is known to be regulated by cell adhesions to basement membranes. In this study differences in anticancer drug-sensitivities of early and advanced endometrial adenocarcinomas were examined by using highly-differentiated human endometrial adenocarcinoma cell line, HHUA, whose apoptotic susceptibility was hypothesized to be modulated by extracellular matrices. The cells express high levels of laminin receptors and laminin suppressed

Fas-mediated apoptosis of the cells. However laminin did not show any effect on apoptotic susceptibility to 4 anticancer drugs. These results suggest that there is little variation in drug-sensitivity associated with disruption of endometrial stromal tissue, and that pathological staging of endometrial carcinoma is not likely to have any relation to drug sensitivity.

CT Check Tags: Female; Human; Support, Non-U.S. Gov't
Adenocarcinoma: PA, pathology
***Adenocarcinoma: PP, physiopathology**
*Antigens, CD95: IM, immunology
*Antineoplastic Agents: PD, pharmacology
*Apoptosis: DE, drug effects
Collagen: PD, pharmacology
Drug Resistance
Endometrial Neoplasms: PA, pathology
*Endometrial Neoplasms: PP, physiopathology
IgM: IM, immunology
***IgM: PD, pharmacology**
***Laminin: PD, pharmacology**
Tumor Cells, Cultured

L170 ANSWER 9 OF 26 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 97168349 EMBASE
DOCUMENT NUMBER: 1997168349
TITLE: Role of sinusoidal heparan sulfate proteoglycan in liver metastasis formation.
AUTHOR: Tovari J.; Paku S.; Raso E.; Pogany G.; Kovalszky I.; Ladanyi A.; Lapis K.; Timar J.
CORPORATE SOURCE: J. Timar, 1st Institute of Pathology, Semmelweis University of Medicine, Ulloi ut 26, H-1085 Budapest, Hungary
SOURCE: International Journal of Cancer, (1997) 71/5 (825-831).
Refs: 24
ISSN: 0020-7136 CODEN: IJCNAW
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
016 Cancer
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Previous studies have indicated that the predominant sites of tumor cell extravasation in the liver are the sinusoidal vessels, where tumor cells contact the sinusoidal endothelium and the subendothelial extracellular matrix containing the basic components of the basement membrane. We studied the role of sinusoidal extracellular matrix in metastatsis formation by 3LL-HH murine tumor cells selected for their preferential liver colonization. 3LL-HH tumor cells did not efficiently adhere to cryosections of the liver, but they recognized the sinusoids and vessel walls. Pre-treatment of the mice with polyclonal anti-basement membrane **antibodies** [anti-**laminin**, anti-fibronectin and anti-heparan sulfate proteoglycan (HSPG)] significantly modulated the organ distribution of tumor cell colonies following intracardial injection: all 3 antibodies inhibited kidney colonization; anti-**laminin** and anti-fibronectin **antibodies** inhibited lung colonization; and only anti-HSPG antibody inhibited liver colonization. In several organs such as the heart, stomach, pancreas and bladder, anti-basement membrane antibody treatment did not alter the process of colonization. Immunofluorescence studies showed that anti-HSPG antibody recognized the basement membranes of sinusoids and blood vessels. Our data suggest a specific involvement of sinusoidal HSPG in the liver

colonization of 3LL-HH cells.
CT Medical Descriptors:
***liver metastasis: DT, drug therapy**
***liver metastasis: PC, prevention**
animal experiment
animal model
animal tissue
article
controlled study
extracellular matrix
immunofluorescence
liver sinusoid
mouse
nonhuman
priority journal
tumor cell
Drug Descriptors:
*proteoheparan sulfate: EC, endogenous compound
monoclonal antibody

L170 ANSWER 10 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1997:440892 BIOSIS
DOCUMENT NUMBER: PREV199799740095
TITLE: Effects of 1,25-dihydroxyvitamin D-3 on tumor cell invasion to the extracellular matrix in human fibrosarcoma HT1080 cells and its correlation with laminin.
AUTHOR(S): Yudoh, Kazuo (1); Matsui, Hisao; Tsuji, Haruo
CORPORATE SOURCE: (1) Dep. Orthop. Surg., Toyama Med. Pharm. Univ., 2630 Sugitami, Toyama 930-01 Japan
SOURCE: Tumor Biology, (1997) Vol. 18, No. 2, pp. 69-79. ISSN: 1010-4283.
DOCUMENT TYPE: Article
LANGUAGE: English
AB We investigated the role of the active form of vitamin D, 1,25-dihydroxyvitamin D-3 (1,25(OH)-2D-3), in promoting tumor cell invasiveness through the extracellular matrix, and showed that 1,25(OH)-2D-3-induced reduction of laminin production by the cells was correlated with the inhibitory effects of the hormone on tumor cell invasiveness. 1,25(OH)-2D-3 significantly **inhibited invasiveness** through the matrix, type IV collagenolytic and migratory activity, but not cell attachment to the matrix in human fibrosarcoma HT1080 cells. The 1,25(OH)-2D-3-induced inhibition showed the same dose dependency and magnitude for invasiveness as for the effects on type IV collagenolysis and cell migration. 1,25(OH)-2D-3 inhibited laminin production from the cells in a dose-dependent manner. The inhibitory effects of 1,25(OH)-2D-3 on the invasiveness, type IV collagenolysis and cell migration appeared to parallel the hormone-induced reduction of **laminin** production. **Antilaminin** monoclonal **antibody**, blocking the activity of **laminin** in the culture medium, **inhibited** HT1080 cell **invasiveness**. In the presence of exogenous laminin, 1,25(OH)-2D-3-induced **inhibition** of **invasion** was not observed. These findings suggest that 1,25(OH)-2D-3 acts on HT1080 cells, inhibiting the expression of laminin from the cells, and that the reduced laminin expression leads to the inhibition in the type IV collagenolytic and migratory activity of the cells, and consequently, to the **inhibition** of **invasiveness** through the extracellular matrix.
IT Major Concepts
Cell Biology; Endocrine System (Chemical Coordination and Homeostasis); Oncology (Human Medicine, Medical Sciences)

IT Chemicals & Biochemicals
1,25-DIHYDROXYVITAMIN D3

L170 ANSWER 11 OF 26 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:534886 HCAPLUS
DOCUMENT NUMBER: 127:232687
TITLE: The relationship between expression of basement membrane in squamous cell carcinoma of oral cavity and cervical lymph node metastasis
AUTHOR(S): Gu, Xiang; Shen, Zihua; Liu, Shufan; Qian, Zhongfei
CORPORATE SOURCE: Dep. Somatol., Xiangya Hosp., Hunan Med. Univ., Changsha, 410008, Peop. Rep. China
SOURCE: Hunan Yike Daxue Xuebao (1997), 22(1), 41-44
CODEN: HYXBET; ISSN: 1000-5625
PUBLISHER: Hunan Yike Daxue
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB 57 Cases of oral squamous cell carcinoma (SCC) were studied by immunohistochem. ABC method using type IV collagen and **laminin antibody** to investigate the relationship between expression and distribution of basement membrane and clinicopathol. characteristics and cervical lymph node metastasis. The distribution of basement membrane of oral SCC was discontinuous and/or partially or completely disappeared. There were highly significant correlation between the staining patterns together with histol. differentiation degrees and cervical lymph node metastasis, $P < 0.05$. The results suggest that the expression of basement membrane in oral SCC is a useful parameter to evaluate the tumor histol. differentiation and tumor invasion and metastasis.
CT Uterus, neoplasm
CT Uterus, neoplasm
CT Lymph node
CT Mouth
CT Mouth
CT Basement membrane
CT **Laminins**
CT **Antibodies**
CT **Carcinoma**
CT Collagens, biological studies

L170 ANSWER 12 OF 26 HCAPLUS COPYRIGHT 2002 ACS DUPLICATE 1
ACCESSION NUMBER: 1996:377252 HCAPLUS
DOCUMENT NUMBER: 125:49293
TITLE: Human laminin 5 .gamma.2-chain antibody for diagnosis and antisense oligonucleotides for inhibition of malignant cell invasive growth
INVENTOR(S): Tryggvason, Karl; Kallunki, Pekka; Pyke, Charles
PATENT ASSIGNEE(S): Finland
SOURCE: PCT Int. Appl., 36 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9610646	A1	19960411	WO 1995-EP3918	19951004

W: AL, AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI,

SK, TJ
RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

US 5660982	A	19970826	US 1994-317450	19941004
CA 2201865	AA	19960411	CA 1995-2201865	19951004
AU 9537451	A1	19960426	AU 1995-37451	19951004
AU 699183	B2	19981126		
EP 784703	A1	19970723	EP 1995-935428	19951004
EP 784703	B1	19990714		

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE

AT 182180	E	19990715	AT 1995-935428	19951004
ES 2133813	T3	19990916	ES 1995-935428	19951004

PRIORITY APPLN. INFO.: US 1994-317450 A 19941004
WO 1995-EP3918 W 19951004

AB The instant invention provides for the identification, diagnosis, monitoring, and treatment of malignant invasive cells using the laminin 5 .gamma.-2 chain protein or nucleic acid sequence, and antibodies or antisense oligonucleotides.

CT Plasmid and Episome
CT Virus, animal
CT Genetic vectors
CT Melanoma
CT Mutation
CT Polymerase chain reaction
CT Sarcoma
CT Gene, animal
CT **Antibodies**
CT Neoplasm
CT **Neoplasm inhibitors**
CT **Laminins**
CT Intestine, neoplasm
CT Skin, disease
CT **Antibodies**
CT Mammary gland
CT Nucleotides, biological studies
CT Nucleotides, biological studies
CT Nucleotides, biological studies
CT **Carcinoma**
CT Skin, disease

L170 ANSWER 13 OF 26 CANCERLIT
ACCESSION NUMBER: 97307465 CANCERLIT
DOCUMENT NUMBER: 97307465
TITLE: Laminin mediates basement membrane induced differentiation of HEC 1B endometrial adenocarcinoma cells.
AUTHOR: Behrens P; Meissner C; Hopfer H; Schumann J; Tan M I; Ellerbrake N; Strunck E; Vollmer G
CORPORATE SOURCE: Institut fur Biochemische Endokrinologie, Medizinische Universitat, Lubeck, Germany.
SOURCE: BIOCHEMISTRY AND CELL BIOLOGY, (1996). Vol. 74, No. 6, pp. 875-86.
Journal code: ALR. ISSN: 0829-8211.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Priority Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 97307465
ENTRY MONTH: 199708

AB In vitro studies on endometrial carcinogenesis have been hampered by limited differentiation of the cells in culture. Using the endometrial

carcinoma cell lines HEC 1B and its subclone HEC 1B(L), we established and characterized cell culture conditions that preserve a more differentiated state of the tumor cells. Randomly seeded HEC 1B(L) cells, if grown in a serum-free defined medium on top of a reconstituted basement membrane (Matrigel), within a few hours assembled themselves to web-like structures. In a thick layer of Matrigel, they showed an even more pronounced morphological differentiation. Functionally, two additional secretory proteins, about 31 and 77 kDa in size, became apparent as a response to matrigel. To further investigate the regulatory role of the extracellular matrix in the process of in vitro differentiation of endometrial adenocarcinoma cells, we addressed two specific problems. First, we investigated if the capacity of in vitro differentiation is a specific feature of HEC 1B(L) cells or if it is common to all endometrial adenocarcinoma cells. Second, we tried to identify the Matrigel component(s) responsible for in vitro differentiation. The assembly of HEC 1B and HEC 1B(L) cells into spatially organized web-like structures and the expression of the 77 kDa protein were thereby used as an assay. All endometrial adenocarcinoma cell lines tested to a variable degree formed web-like structures on Matrigel. Although the pattern of de novo synthesized secretory proteins changed as a response to Matrigel, only HEC 1A, HEC 1B, HEC 1B(L), and Ishikawa cells responded to culture on Matrigel by an increased expression of the 77 kDa protein. Functionally, polyclonal anti-laminin antibodies, but not anti-collagen type IV antibodies, disrupted formation of web-like structures by HEC 1B cells. The laminin-specific peptides YIGSR and SIKVAV but none of the RGD-peptides RGDS, GRGDSP, or GRADSP affected the three-dimensional assembly of these cells in vitro. Both anti-laminin antibodies and laminin-specific peptides suppressed Matrigel-induced formation of the 77-kDa secretory protein by HEC 1B cells. These findings suggest the involvement of laminin in the in vitro differentiation of the HEC 1B endometrial adenocarcinoma cell line. In a mechanistic view, laminin appears to play a crucial role in the regulation of this in vitro differentiation process.

CT Check Tags: Female; Human; Support, Non-U.S. Gov't
***Adenocarcinoma: PA, pathology**
Antibodies: PD, pharmacology
*Basement Membrane: PH, physiology
*Cell Differentiation
Collagen: AI, antagonists & inhibitors
Collagen: PH, physiology
Culture Media
Drug Combinations
*Endometrial Neoplasms: PA, pathology
Laminin: AI, antagonists & inhibitors
***Laminin: PH, physiology**
Peptide Fragments: PD, pharmacology
Proteoglycans
Tumor Cells, Cultured

L170 ANSWER 14 OF 26 CANCERLIT
ACCESSION NUMBER: 97092076 CANCERLIT
DOCUMENT NUMBER: 97092076
TITLE: Reduction of LAK-sensitivity and changes in antigen expression on hepatoma cells by sodium butyrate.
AUTHOR: Tada S; Saito H; Ebinuma H; Atsukawa K; Masuda T; Tsunematsu S; Morizane T; Ishii H
CORPORATE SOURCE: Department of Internal Medicine, School of Medicine, Keio University, Tokyo, Japan.
SOURCE: CANCER BIOCHEMISTRY BIOPHYSICS, (1996). Vol. 15, No. 3, pp. 177-86.
Journal code: CL0. ISSN: 0305-7232.

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Priority Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 97092076
ENTRY MONTH: 199704

AB We demonstrated that sodium butyrate (SB) induced differentiation of functions in human hepatocellular carcinoma (HCC) cell lines. To investigate relationship between the sensitivity for cellular cytotoxicity and the cellular differentiation of HCC cells, the effect of SB on lymphokine-activated killer (LAK) sensitivity and antigen expression of a human HCC cells were studied. SB induced LAK-resistance of human HCC cell lines, HCC-T and HCC-M, time-dependently. A flowcytometric analysis of cell surface antigens revealed that SB markedly reduced the expression of laminin and fibronectin and increased the expression of liver-specific antigen defined by a mouse monoclonal antibody time-dependently, but did not modify that of major histocompatibility complex antigens, intercellular adhesion molecule (ICAM)-1, or CEA. Leukocyte function-associated antigen (LFA)-3 expression on HCC-T was reduced slightly by SB treatment. LAK sensitivity was inhibited by **anti-laminin**, but not with **anti**-beta 2-microglobulin, anti-HLA DR, anti-ICAM-1, anti-fibronectin, or anti-CEA. Anti-LFA-3 reduced LAK sensitivity of HCC-T, but not HCC-M, although the reduction was less than that obtained by **anti-laminin** treatment. These results provided evidence that SB induced LAK-resistance of human HCC cells according to cellular differentiation and extracellular matrix functionality played an important role in this LAK-mediated cell killing. Moreover, the structure expressed on HCC cells, which contributed to LAK cytolysis, was different for each HCC cell.

CT Check Tags: Animal; Human; Support, Non-U.S. Gov't
Antibodies, Monoclonal: PD, pharmacology
*Antigens, Neoplasm: BI, biosynthesis
Antigens, Neoplasm: IM, immunology
Antigens, Surface: BI, biosynthesis
*Butyric Acids: PD, pharmacology
***Carcinoma, Hepatocellular: DT, drug therapy**
*Carcinoma, Hepatocellular: ME, metabolism
Carcinoma, Hepatocellular: PA, pathology
Cell Communication: DE, drug effects
Cell Differentiation: DE, drug effects
Gap Junctions: DE, drug effects
*Killer Cells, Lymphokine-Activated: DE, drug effects
Killer Cells, Lymphokine-Activated: IM, immunology
*Liver Neoplasms: DT, drug therapy
*Liver Neoplasms: ME, metabolism
Liver Neoplasms: PA, pathology
Mice
Sensitivity and Specificity
Tumor Cells, Cultured: DE, drug effects

L170 ANSWER 15 OF 26 CANCERLIT
ACCESSION NUMBER: 96121214 CANCERLIT
DOCUMENT NUMBER: 96121214
TITLE: Human trophoblast adhesion to matrix proteins: inhibition and signal transduction.
AUTHOR: Burrows T D; King A; Smith S K; Loke Y W
CORPORATE SOURCE: Department of Pathology, University of Cambridge, UK.
SOURCE: HUMAN REPRODUCTION, (1995). Vol. 10, No. 9, pp. 2489-500. Journal code: HRP. ISSN: 0268-1161.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Priority Journals

LANGUAGE: English
OTHER SOURCE: MEDLINE 96121214
ENTRY MONTH: 199603

AB At the time of implantation, the extracellular matrix proteins laminin and fibronectin are abundant in the decidua and are distributed pericellularly around each individual stromal cell. First trimester human trophoblast expresses both laminin and fibronectin receptors, specifically the alpha 1 beta 1, alpha 5 beta 1, alpha 6 beta 1 and alpha 6 beta 4 integrin heterodimers. In this study we have demonstrated that in-vitro adhesion of first trimester human trophoblast to purified extracellular matrix proteins and to purified decidual stromal cell monolayers can be inhibited by monoclonal antibodies directed against appropriate integrin subunits and by synthetic peptides containing an arginine-glycine-aspartic acid sequence. Monoclonal antibodies (mAbs) to the alpha 5 and beta 1 integrin subunits and a synthetic peptide significantly inhibited adhesion to fibronectin. Binding of trophoblast to laminin was blocked with mAbs to the alpha 6 and beta 1 but not alpha 1 and beta 4 integrin subunits. Similarly, integrin-mediated adhesion to monolayers of decidual stromal cells could be blocked with mAbs to the alpha 5, alpha 6, beta 1 and beta 4 integrin subunits. Integrin-mediated signal transduction in normal and malignant trophoblast was investigated by Western blotting. A 115 kDa protein was the major tyrosine phosphorylated protein detected in trophoblast after binding to laminin or fibronectin. The profile of tyrosine phosphorylated proteins differed for malignant trophoblast.

CT Check Tags: Animal; Female; Human; Support, Non-U.S. Gov't
Amino Acid Sequence
Antibodies, Monoclonal: PD, pharmacology
Cell Adhesion
Choriocarcinoma
Decidua: CY, cytology
*Fibronectins: ME, metabolism
Integrins: IM, immunology
*Integrins: PH, physiology
***Laminin: ME, metabolism**
Mice
Molecular Sequence Data
Peptides: PD, pharmacology
Pregnancy
*Signal Transduction
Stromal Cells: CY, cytology
*Trophoblast: ME, metabolism
Tumor Cells, Cultured
3T3 Cells

L170 ANSWER 16 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 2
ACCESSION NUMBER: 1995:300775 BIOSIS
DOCUMENT NUMBER: PREV199598315075
TITLE: Inhibition of experimental **metastasis** of human breast carcinoma cells in athymic nude mice by anti-alpha-5-beta-1 fibronectin receptor integrin antibodies.
AUTHOR(S): Newton, Sheila A.; Reeves, Emily J.; Gralnick, Harvey; Mohla, Suresh; Yamada, Kenneth M.; Olden, Kenneth; Akiyama, Steven K. (1)
CORPORATE SOURCE: (1) Lab. Dev. Biol., Natl. Inst. Dent. Res., Building 30, Room 421, Natl. Inst. Health, Bethesda, MD 20892-4370 USA
SOURCE: International Journal of Oncology, (1995) Vol. 6, No. 5, pp. 1063-1070.
ISSN: 1019-6439.

DOCUMENT TYPE: Article
LANGUAGE: English
AB We have investigated the role of the human alpha-5-beta-1 fibronectin receptor integrin in experimental **metastasis**. Treatment of human MDA-MB-231 breast carcinoma cells with monoclonal antibodies specific for alpha-5 or beta-1 integrin subunits prior to injection into the tail veins of 7 to 9 week old athymic nude mice significantly decreased the median number of lung colonies that were formed. In contrast, treatment of the cells with monoclonal antibodies specific for the alpha-2 subunit had no significant effect. In vitro, the anti-alpha-5 and the anti-beta-1 monoclonal antibodies both strongly **inhibited** breast **carcinoma** cell adhesion to fibronectin, while only the anti-beta-1 monoclonal **antibody** inhibited adhesion to **laminin**. In a Boyden chamber **invasion** assay, the anti-beta-1 antibody almost completely **inhibited invasion** of the breast **carcinoma** cells through an artificial Matrigel basement membrane. The anti-a, monoclonal antibody inhibited in vitro **invasion** approximately 30%, only if fibroblast conditioned medium was present as a chemoattractant. Cell migration on fibronectin could be inhibited by both the anti-alpha-5 and the anti-beta-1 monoclonal antibody. These results indicate that the alpha-5,beta-1 integrin fibronectin receptor on MDA-MB-231 human breast carcinoma cells plays an important role in experimental hematogenous **metastasis** and may function in this process by a combination of mechanisms, including tumor cell attachment to fibronectin and fibronectin-directed extravasation of tumor cells into the target organ.
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Cell Biology; Immune System (Chemical Coordination and Homeostasis); Membranes (Cell Biology); Oncology (Human Medicine, Medical Sciences); Reproductive System (Reproduction)
IT Chemicals & Biochemicals
INTEGRIN

L170 ANSWER 17 OF 26 CANCERLIT
ACCESSION NUMBER: 96007602 CANCERLIT
DOCUMENT NUMBER: 96007602
TITLE: Rapid spreading and mature hemidesmosome formation in HaCaT keratinocytes induced by incubation with soluble laminin-5r.
AUTHOR: Hormia M; Falk-Marzillier J; Plopper G; Tamura R N; Jones J C; Quaranta V
CORPORATE SOURCE: Department of Cell Biology, Scripps Research Institute, La Jolla, California 92037, USA.
CONTRACT NUMBER: TW04710 (FIC)
GM46902 (NIGMS)
DE10063 (NIDCR)
SOURCE: JOURNAL OF INVESTIGATIVE DERMATOLOGY, (1995). Vol. 105, No. 4, pp. 557-61.
Journal code: IHZ. ISSN: 0022-202X.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Priority Journals; Cancer Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 96007602
ENTRY MONTH: 199512
AB HaCaT cells, an immortalized keratinocyte line, incubated in plastic wells in the presence of conditioned medium from 804G cells adhered and spread rapidly in less than 30 min. In contrast, cells plated in fibroblast or keratinocyte conditioned medium adhered poorly and remained rounded at 30 min. Immunodepletion of 804G conditioned medium with polyclonal antisera

to laminin-5r, but not control antisera, abolished rapid cell spreading. Electron microscopy of HaCaT cells spread by incubation in 804G conditioned medium, but not control medium, revealed mature hemidesmosomes after 24 h. Rapid spreading was also observed in wells precoated with 804G conditioned medium or 804G cell-deposited matrix, but not with fibronectin, vitronectin, or laminin-1. Immunoblotting of 804G conditioned medium with anti-laminin-5r antibodies unveiled polypeptides of 150, 140, 135, and 100 kDa, identical by electrophoretic mobility to immunoreactive polypeptides in 804G deposited matrix. Our results suggest that addition of laminin-5r in a soluble form is sufficient to promote rapid spreading and hemidesmosome assembly in keratinocytes. The mechanism of soluble laminin-5r action may include efficient surface "priming" for cell adhesion. Soluble laminin-5r may have a physiologic role in morphogenesis and repair of the epidermis and may be of use for therapeutic applications.

CT Check Tags: Animal; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.
Bladder Neoplasms: PA, pathology
Carcinoma: PA, pathology
Cell Adhesion: PH, physiology
Cell Line, Transformed
Cell Size
Culture Media, Conditioned: PD, pharmacology
Extracellular Matrix Proteins: PD, pharmacology
Immune Sera: PD, pharmacology
*Intercellular Junctions: ME, metabolism
*Keratinocytes: DE, drug effects
Keratinocytes: ME, metabolism
Keratinocytes: UL, ultrastructure
***Laminin: PD, pharmacology**
*Neoplasm Proteins: PD, pharmacology
Rats
Solubility
Tumor Cells, Cultured
Wound Healing

L170 ANSWER 18 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1995:85887 BIOSIS
DOCUMENT NUMBER: PREV199598100187
TITLE: Integrin switching regulates normal trophoblast invasion.
AUTHOR(S): Damsky, Caroline H. (1); Librach, Clifford; Lim, Kee-Hak; Fitzgerald, Marilyn L.; McMaster, Michael T.; Janatpour, Mary; Zhou, Yan; Logan, Susan K.; Fisher, Susan J.
CORPORATE SOURCE: (1) Dep. Stomatol., Univ. Calif. San Francisco, San Francisco, CA 94143-0512 USA
SOURCE: Development (Cambridge), (1994) Vol. 120, No. 12, pp. 3657-3666.
ISSN: 0950-1991.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Cells invade extracellular matrices in a regulated manner at specific times and places during normal development. A dramatic example is trophoblast invasion of the uterine wall. Previous studies have shown that differentiation of trophoblasts to an invasive phenotype is accompanied by temporally and spatially regulated switching of their integrin repertoire. In the first trimester human placenta, alpha-6 integrins are restricted to cytotrophoblast (CTB) stem cells and downregulated in invasive CTBs, whereas alpha-5-beta-1 and alpha-1-beta-1 integrins are upregulated in differentiating and invasive CTBs. The goal of the present study was to determine whether these changes have functional consequences for CTB

invasiveness. Using an in vitro invasion model, we determined first that aggregates of invading first trimester CTBs in vitro undergo the same pattern of integrin switching as was observed in situ, thereby validating the utility of the model. We then showed that **antibody perturbation** of interactions involving **laminin** or collagen type IV and their integrin alpha-1/beta-1 receptor **inhibited invasion** by CTBs, whereas perturbing interactions between fibronectin and the alpha-5/beta-1 fibronectin receptor accelerated invasion. Finally, we report that later gestation CTBs, which display greatly decreased invasive capacity, are also unable to upregulate alpha-1-beta-1 complexes, providing further evidence that this integrin is critical for CTB invasion. This gestational regulation is transcriptional. These data indicate that integrin switching observed during differentiation in situ has significant functional consequences for CTB invasion. The data suggest further that differentiating CTBs upregulate counterbalancing invasion-accelerating and invasion-restraining adhesion mechanisms. We propose that this contributes to regulating the depth of CTB invasion during normal implantation.

IT Major Concepts
Development; Membranes (Cell Biology); Reproductive System (Reproduction)

IT Chemicals & Biochemicals
INTEGRIN

L170 ANSWER 19 OF 26 CANCERLIT

ACCESSION NUMBER: 94252732 CANCERLIT
DOCUMENT NUMBER: 94252732
TITLE: Laminin receptor expression and function in small-cell lung carcinoma.
AUTHOR: Pellegrini R; Martignone S; Menard S; Colnaghi M I
CORPORATE SOURCE: Division of Experimental Oncology E, Istituto Nazionale Tumori, Milan, Italy.
SOURCE: INTERNATIONAL JOURNAL OF CANCER. SUPPLEMENT, (1994). Vol. 8, pp. 116-20.
Journal code: GRM. ISSN: 0020-7136.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Priority Journals; Cancer Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 94252732
ENTRY MONTH: 199408

AB Interactions between tumor cells and laminin or other components of the extracellular matrix are thought to play a role in tumor invasion and metastasis. To analyze these interactions, we examined the expression of 5 types of laminin receptors on 11 cell lines derived from the highly malignant and metastatic tumor small-cell lung cancer (SCLC). The integrins VLA-1, VLA-3, VLA-6 and the 67 KDa monomeric receptor were expressed at various levels, whereas the VLA-2 receptor was absent on the cell lines. Only one cell line expressed none of these laminin receptors. All cell lines co-expressed alpha 6 beta 1 (VLA-6) and the 67-kDa molecule, the only receptors specific for laminin. Analysis of the ability of SCLC cells to bind radiolabeled laminin and to adhere to laminin substrata revealed a correlation between these 2 parameters and the expression of VLA-6 and the 67-kDa monomeric receptor. Cell adhesion was mediated by alpha 6 beta 1, as indicated by inhibition of adhesion using an anti-VLA-6 monoclonal antibody (MAb). Both VLA-6 and the monomeric receptor were up-regulated in vitro by laminin.

CT Check Tags: Human; Support, Non-U.S. Gov't
Antibodies: PD, pharmacology
Antibodies, Monoclonal: PD, pharmacology
***Carcinoma, Small Cell: ME, metabolism**

Cell Adhesion: DE, drug effects
Cell Line
Fluorescent Antibody Technique
*Integrins: BI, biosynthesis
Integrins: DE, drug effects
Integrins: ME, metabolism
***Laminin: ME, metabolism**
*Lung Neoplasms: ME, metabolism
Molecular Weight
*Receptors, Laminin: BI, biosynthesis
Receptors, Laminin: ME, metabolism
Regression Analysis
Tumor Cells, Cultured

L170 ANSWER 20 OF 26 CANCERLIT
ACCESSION NUMBER: 93179246 CANCERLIT
DOCUMENT NUMBER: 93179246
TITLE: Clinical significance of laminin deposition and T-cell infiltration in oral cancer.
AUTHOR: Noguchi M; Kohama G; Hiratsuka H; Sekiguchi T
CORPORATE SOURCE: Department of Oral Surgery, Sapporo Medical College, Japan.
SOURCE: HEAD AND NECK, (1993). Vol. 15, No. 2, pp. 125-32. Journal code: G1P. ISSN: 1043-3074.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Priority Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 93179246
ENTRY MONTH: 199305

AB Biopsied specimens from 55 patients with squamous cell carcinoma (SCC) of the oral cavity were examined immunohistopathologically as to the clinical significance of basement membrane (BM) deposition and T-cell infiltration at the tumor-stromal border using monoclonal **anti-laminin** and **anti**-CD3 **antibodies**. According to the immunoreactivity, all specimens could be divided into three groups: group A, a continuous linear pattern of positive staining for BM around tumor nests; group B, an alteration of BM deposition around tumor nests with T cell infiltration into those tumor nests; and group C, an alteration of BM deposition around tumor nests without T cell infiltration into those tumor nests. These groups were correlated with clinical manifestations, such as tumor size, tumor regression rate with induction chemotherapy, and regional lymph node metastatic rate. In these groups, tumors classified as group C showed a trend toward resistance to chemotherapy and high metastatic characteristics. Tumors classified as group B, which showed the same alteration of BM deposition as a result of T cell infiltration into the tumor nests, showed a sufficient tumor regression rate with chemotherapy. The visualization of the staining for BM laminin and T cells in oral SCC appeared not only to increase our understanding of the biologic and clinical behavior of individual tumors, but could be a prognostic indicator.

CT Check Tags: Human; Support, Non-U.S. Gov't
Antibodies, Monoclonal
Antineoplastic Agents, Combined: TU, therapeutic use
Basement Membrane: ME, metabolism
Basement Membrane: PA, pathology
Carcinoma, Squamous Cell: DT, drug therapy
*Carcinoma, Squamous Cell: ME, metabolism
*Carcinoma, Squamous Cell: PA, pathology
Immunoenzyme Techniques
*Laminin: ME, metabolism
Lymphatic Metastasis: PA, pathology

Mouth Mucosa: ME, metabolism
Mouth Mucosa: PA, pathology
Mouth Neoplasms: DT, drug therapy
*Mouth Neoplasms: ME, metabolism
*Mouth Neoplasms: PA, pathology
Neoplasm Staging
Remission Induction
*T-Lymphocytes: PA, pathology

L170 ANSWER 21 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 3
ACCESSION NUMBER: 1992:7193 BIOSIS
DOCUMENT NUMBER: BA93:7193
TITLE: LAMININ RECEPTOR COMPLEMENTARY DNA-DEDUCED SYNTHETIC PEPTIDE INHIBITS CANCER CELL ATTACHMENT TO ENDOTHELIUM.
AUTHOR(S): CASTRONOVO V; TARABOLETTI G; SOBEL M E
CORPORATE SOURCE: LAB. PATHOL., NATL. CANCER INST., BUILD. 10, ROOM 2A33, BETHESDA, MD. 20892.
SOURCE: CANCER RES, (1991) 51 (20), 5672-5678.
CODEN: CNREA8. ISSN: 0008-5472.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB Stable attachment of cancer cells to the endothelium is a key step in the formation of metastasis. In this study, we have investigated the possibility that interaction between laminin and its Mr 67,000 high-affinity receptor (67 LR) could play a major role in this process. Scatchard analysis of laminin-binding studies showed that bovine aortic endothelial cells exhibit 46,000 high-affinity receptors that mediate, at least in part, the attachment of highly invasive melanoma cells. This endothelial cell-melanoma cell interaction was significantly inhibited by soluble **laminin** and by **anti-laminin antibodies**. Peptide G, an eicosapeptide derived from the complementary DNA sequence of the 67 LR precursor (IPCNNKGAHSVGLMWWMLAR) that specifically binds to laminin and presumably contains the active ligand-binding site of the receptor, specifically prevented attachment of the melanoma cells to both the bovine aortic endothelial cell monolayer and human umbilical vein endothelium. Thus, peptide G may selectively interfere with the **metastatic** cascade by **inhibiting** tumor cell attachment to endothelium via the laminin-67 LR pathway and is a potential new antimetastatic agent.

L170 ANSWER 22 OF 26 CANCERLIT
ACCESSION NUMBER: 91299805 CANCERLIT
DOCUMENT NUMBER: 91299805
TITLE: Human colon carcinoma cells use multiple receptors to adhere to laminin: involvement of alpha 6 beta 4 and alpha 2 beta 1 integrins.
AUTHOR: Lotz M M; Korzelius C A; Mercurio A M
CORPORATE SOURCE: Laboratory of Cancer Biology, New England Deaconess Hospital, Harvard Medical School, Boston, MA 02115.
CONTRACT NUMBER: CA44704 (NCI)
SOURCE: CELL REGULATION, (1990). Vol. 1, No. 3, pp. 249-57.
Journal code: A1U. ISSN: 1044-2030.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Priority Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 91299805
ENTRY MONTH: 199109
AB In this study, we used clone A, a human colon carcinoma cell line, to characterize those integrins that mediate colon carcinoma adhesion to

laminin. Monoclonal antibodies specific for the human beta 1 subunit inhibited clone A adhesion to laminin. They also precipitated a complex of surface proteins that exhibited an electrophoretic behavior characteristic of alpha 2 beta 1 and alpha 3 beta 1. A monoclonal antibody specific for alpha 2 (PIH5) blocked clone A adhesion to laminin, as well as to collagen I. An alpha 3-specific antibody (P1B5) had no effect on clone A adhesion to laminin, even though it can block the adhesion of other cell types to laminin. Thus, the alpha 2 beta 1 integrin can function as both a laminin and collagen I receptor on clone A cells. Although these cells express alpha 3 beta 1, an established laminin receptor, they do not appear to use it to mediate laminin adhesion. In addition, the monoclonal antibody GoH3, which recognizes the alpha 6 integrin subunit, also inhibited carcinoma adhesion to laminin but not to fibronectin or collagen I. This antibody precipitated the alpha 6 subunit in association with the beta 4 subunit. There was no evidence of alpha 6 beta 1 association on these cells. In summary, the results obtained in this study indicate that multiple integrin alpha subunits, in association with two distinct beta subunits, are involved in colon carcinoma adhesion to laminin. Based on the behavior of alpha 3 beta 1 and alpha 2 beta 1, the results also suggest that cells can regulate the ability of a specific integrin to mediate adhesion.

CT Check Tags: Comparative Study; Human; Support, U.S. Gov't, P.H.S.
***Adenocarcinoma: PA, pathology**
Antibodies, Monoclonal: PD, pharmacology
*Cell Adhesion
Cell Differentiation
Collagen: ME, metabolism
*Colonic Neoplasms: PA, pathology
Fibronectins: ME, metabolism
Integrins: IM, immunology
*Integrins: PH, physiology
***Laminin: ME, metabolism**
Tumor Cells, Cultured: ME, metabolism
Tumor Cells, Cultured: PA, pathology

L170 ANSWER 23 OF 26 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1990:569641 HCAPLUS
DOCUMENT NUMBER: 113:169641
TITLE: Immunohistochemical studies on laminin in gastric cancers and metastatic lesions
AUTHOR(S): Sakaguchi, Takahiro
CORPORATE SOURCE: Sch. Med., Kinki Univ., Osaka, Japan
SOURCE: Kinki Daigaku Igaku Zasshi (1989), 14(3), 537-51
CODEN: KDIZDD; ISSN: 0385-8367
DOCUMENT TYPE: Journal
LANGUAGE: Japanese

AB An immunohistol. study of laminin in human gastric cancers was performed. Hepatic metastasis were also examd. Immunohistol. staining was performed with rabbit anti-human **laminin antibody** as the primary antibody and by the ABC method. The pos. response rate for gastric cancer was 23.4%. According to histol. type, the pos. response rate was 46.7% for well differentiated adenocarcinomas, 29.3% for moderately differentiated ones and only 13.0% for poorly differentiated ones, the higher the degree of differentiation, the higher the rate. When poorly differentiated adenocarcinomas were classified newly into solid, medullary and scirrhous types, all the 6 laminin-pos. cases were of the solid type. The pos. response rate was 14.7% in cases without hepatic metastasis, 88.9% in cases complicated with hepatic metastasis and 91.7% for hepatic metastatic lesions, the rate being higher in cases of hepatic metastasis. In poorly differentiated adenocarcinomas 3 out of 4 cases complicated with hepatic metastasis were pos. for laminin and were of the solid type. As a

result, laminin-pos. cancers showed hematogenous metastasis by participating in the mass formation of cancer cells and in the attachment of the cancer cells to the basement membrane in the target organs during the process of hematogenous metastasis. It is further suggested that the conventional relationship between the histol. type and hepatic metastasis can be supported from the standpoint of laminin stain.

CT Laminins
CT **Carcinoma**
CT Stomach, neoplasm
CT Liver, neoplasm

L170 ANSWER 24 OF 26 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 1987-170334 [24] WPIDS
DOC. NO. CPI: C1987-070984
TITLE: Recombinant DNA clone encoding **laminin** receptor - used in diagnostic assays for cancer cells and for producing peptide(s) to **inhibit** cancer **metastases**.
DERWENT CLASS: B04 D16
INVENTOR(S): DROHAN, W N; JAYE, M C; LIOTTA, L A; SOBEL, M E; WEWER, U M
PATENT ASSIGNEE(S): (RORE) RORER INT HOLDINGS INC; (USDC) US DEPT OF COMMERCE; (USSH) US DEPT HEALTH & HUMAN SERVICE; (USDC) US SEC OF COMMERCE; (RHON) RHONE POULENC RORER INT HOLDINGS INC; (USGO) US GOVERNMENT
COUNTRY COUNT: 14
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 911863	A0	19870324	(198724)*		48
WO 8802407	A	19880407	(198815)	EN	
RW: AT BE CH DE FR GB IT LU NL SE					
W: JP					
EP 324788	A	19890726	(198930)	EN	
R: AT BE CH DE FR GB IT LI LU NL SE					
US 4861710	A	19890829	(198944)		18
JP 02500637	W	19900308	(199016)		
CA 1305678	C	19920728	(199236)		
EP 561172	A1	19930922	(199338)	EN	22
R: AT BE CH DE FR GB IT LI LU NL SE					
JP 06107691	A	19940419	(199420)		18
EP 324788	B1	19940706	(199426)	EN	23
R: AT BE CH DE FR GB IT LI LU NL SE					
DE 3750180	G	19940811	(199431)		
EP 324788	A4	19900516	(199511)		
JP 08080200	A	19960326	(199622)		17
JP 2533595	B2	19960911	(199641)		18
JP 2825780	B2	19981118	(199851)		20

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
US 911863	A0		US 1986-911863	19860926
EP 324788	A		EP 1987-907048	19870923
JP 02500637	W		JP 1987-506395	19870923
CA 1305678	C		CA 1987-548000	19870928
EP 561172	A1	Related to	EP 1987-907048	19870923
			EP 1993-102626	19870923

JP 06107691	A	Div ex	JP 1987-506395	19870923
			JP 1993-116331	19870923
EP 324788	B1		EP 1987-907048	19870923
			WO 1987-US2411	19870923
DE 3750180	G		DE 1987-3750180	19870923
			EP 1987-907048	19870923
			WO 1987-US2411	19870923
EP 324788	A4		EP 1987-907048	
JP 08080200	A	Div ex	JP 1987-506395	19870923
			JP 1995-248671	19870923
JP 2533595	B2		JP 1987-506395	19870923
			WO 1987-US2411	19870923
JP 2825780	B2	Div ex	JP 1987-506395	19870923
			JP 1995-248671	19870923

FILING DETAILS:

PATENT NO	KIND		PATENT NO
EP 324788	B1	Based on	WO 8802407
DE 3750180	G	Based on	EP 324788
		Based on	WO 8802407
JP 2533595	B2	Previous Publ.	JP 02500637
		Based on	WO 8802407
JP 2825780	B2	Previous Publ.	JP 08080200

PRIORITY APPLN. INFO: US 1986-911863 19860926

AB US N6911863 N UPAB: 20011211

Novel recombinant cDNA clone encodes high affinity (10 power-8 to 10 power-10 Kd) cell surface receptors for **laminin**. The CDNA clone may be prepd. by (a) synthesising cDNA using mRNA template from human umbilical vein endothelial cells, (b) inserting endothelial cell cDNA into the lambda gt 11 vector (ATCC 37194), (c) packaging the recombinant lambda gt 11 and using it to infect E. coil 1090 cells (ATCC 37197), (d) screening the resulting lambda gt 11 human endothelial cell cDNA expression library with a monoclonal **antibody** which recognises a domain of the human **laminin** receptor involved in binding of **laminin**, (e) analysing the cDNA inserts by restriction endonuclease mapping to find a common domain and (f) subcloning the largest cDNA insert of the purified phage into plasmid to facilitate further analysis and DNA sequencing.

USE - The synthetic peptides generated from the cDNA sequence can be used to inhibit cancer **metastases**. The **laminin** receptor cDNA can also be used in diagnositic assays for cells contg. **laminin** receptor mRNA, such as human cancer cells, for diagnosis and prognosis of cancer. It can also be used in diagnostic assays for cells contg. altered **laminin** receptor DN.

Dwg.0/0

L170 ANSWER 25 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 4

ACCESSION NUMBER: 1988:135012 BIOSIS
DOCUMENT NUMBER: BA85:69839
TITLE: LOCALIZATION OF A TUMOR CELL ADHESION DOMAIN OF **LAMININ** BY A MONOCLONAL **ANTIBODY**.
AUTHOR(S): SKUBITZ A P N; CHARONIS A S; TSILIBARY E C; FURCHT L T
CORPORATE SOURCE: DEP. LAB. MED. AND PATHOL., UNIV. MINNESOTA, MINNEAPOLIS, MINN. 55455.
SOURCE: EXP CELL RES, (1987) 173 (2), 349-369.
CODEN: ECREAL. ISSN: 0014-4827.

FILE SEGMENT: BA; OLD
LANGUAGE: English

AB Monoclonal antibodies were prepared to localize the domain(s) of laminin to which tumor cells adhere. Rat Y3-Ag 1.2.3 myeloma cells were fused with spleen cells from a rat immunized with a purified 440-kDa fragment of chymotrypsin-digested **laminin**. Three monoclonal **antibodies** (AL-1 to AL-3) that bound to intact laminin in a solid-phase radioimmunoassay were chosen for further analysis. The epitopes recognized by these antibodies were characterized by radioimmunoassays, immunoblotting, radioimmunoprecipitation, and immunoaffinity chromatography. In cell adhesion assays, monoclonal antibody AL-2 **inhibited** the binding of the highly **metastatic** melanoma cell line, K-1735-M4, to both intact laminin and the 440-kDa fragment of laminin. Electron microscopic examination of **laminin-monoclonal antibody** interactions showed that monoclonal antibody AL-2 reacted with the long arm of laminin directly below the cross-region. Two monoclonal antibodies that failed to inhibit tumor cell adhesion to laminin reacted with epitopes on the lateral short arms or cross-region of laminin as seen by electron microscopy. These results suggest that a new tumor cell binding domain of laminin may be located close to the cross-region on the long arm of laminin.

L170 ANSWER 26 OF 26 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 1984-263458 [42] WPIDS
DOC. NO. NON-CPI: N1984-196949
DOC. NO. CPI: C1984-111605
TITLE: Cell receptor for **laminin** and fragments obtd. enzymatically - useful in diagnosis and **treatment** of cancers.
DERWENT CLASS: B04 D16 J04 S03
INVENTOR(S): LIOTTA, L A; RAO, N; TERRANOVA, V P
PATENT ASSIGNEE(S): (USSH) US DEPT HEALTH & HUMAN SERVICE; (USGO) US GOVERNMENT; (USDC) US SEC OF COMMERCE
COUNTRY COUNT: 4
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 8403946	A	19841011	(198442)*	EN	41
W: FI JP					
JP 60500974	W	19850627	(198532)		
FI 8404753	A	19841203	(198537)		
US 4565789	A	19860121	(198606)		
CA 1245153	A	19881122	(198851)		
JP 06207940	A	19940726	(199434)		12
JP 06219961	A	19940809	(199436)		12
JP 06219965	A	19940809	(199436)		12

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 8403946	A		WO 1984-US498	19840404
JP 60500974	W		JP 1984-501642	19840404
US 4565789	A		US 1983-481934	19830404
JP 06207940	A	Div ex	JP 1984-501642	19840404
			JP 1993-291328	19840404
JP 06219961	A	Div ex	JP 1984-501642	19840404
			JP 1993-291326	19840404

JP 06219965 A Div ex JP 1984-501642 19840404
JP 1993-291327 19840404

PRIORITY APPLN. INFO: US 1983-481934 19830404
AB WO 8403946 A UPAB: 19930925
Cell receptor for **laminin** (I) is novel. (I) fragment having a binding domain for the cell receptor defined above and lacking a binding domain for type IV collagen is novel. Alpha 3 (I) fragment obtd. by digestion of (I) with alpha-thrombin is novel.

USE/ADVANTAGE - The cell receptor for (I) has a high affinity for receptor binding domains in the (I) molecule and is characteristic of human cancer cells and perhaps epithelial cells. The receptor is present on the surface of these cells and can be isolated from plasma membrane extracts. Assays are applied to clinical specimens for the detection and quantification of (I) cell receptors expressed as **metastatasising** cells or for the isolation of highly **metastatic** tumour cells from a mixed population. (I) fragments are applied to hosts to block attachment of tumour cells to type IV collagen and to reduce haematogenous formation of **metastases**. The fragments may also be used as ligands for known chemotherapeutic agents, e.g. toxins, so that the conjugates may directly introduced for treatment of cancers, and for drug evaluation in vitro and in vivo, and for the evaluation of synthetic binding sites etc. The (I) and its fragments act as growth factor on receptive cells, to promote cell attachment and dispersion and to stimulate cell division.
0/2